

**Differentiation of mechanism and prognosis of
traumatic brain stem lesions detected by
magnetic resonance imaging in the acute stage**

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SUMMARY

We obtained MRI in 17 patients with primary brain stem injury in the acute stage. Clinical and radiological findings were analyzed in these 17 patients. T2-weighted imaging proved to be most sensitive and specific for the diagnosis of primary brain stem injury. From the MR imaging, we found two different pattern of brain stem injury. A focal brain stem injury which is thought to be due to direct impact has good prognosis and brain stem injury probably associated with diffuse axonal injury has poor prognosis. These acute stage findings are seen only temporally so that it is most important to examine MRI findings in the acute stage to evaluate the prognosis of the patient. The MRI was valuable in predicting outcome and understanding the mechanism of primary brain stem injury.

KEY WORDS

ABR, brain stem, diffuse axonal injury, head injury, MRI

INTRODUCTION

Immediate impact injury of the brain stem has been considered to be primary brain stem injury, and this was differentiated from secondary brain stem injury due to complications of cerebral herniation or hypoxic damage(6). Studies involving autopsy cases or computed tomographic (CT) scan findings of primary brain stem injury have been reported by several authors(1,2,9,11). And some magnetic resonance imaging(MRI) studies of the patients with head injury were reported(10,12). However, clinical studies involving of MRI for primary brain stem injury in acute stage are scant(3,9).

The concepts of mechanically induced damage to the brain stem include lesions produced by diffuse shearing forces and by direct impact. Previous pathological study showed primary brain stem injury is always accompanied by supratentorial cerebral diffuse axonal injury (9). Another mechanism is direct impact of brain stem on the free edge of the tentorium (2,11). These brain stem injury with diffuse axonal injury and direct brain stem injury without axonal injury has not been yet established clinically and radiologically. Different mechanisms of damage may produce lesions in different locations. We have used MR imaging in our head injured patients to assess if the findings indicate distinct patterns of lesions with

different associations and we attempted to establish a relationship between MRI findings and clinical course and findings obtained with other radiological or electrophysiological examinations.

CLINICAL MATERIAL AND METHODS

Study population

From 1987 until 1992, 74 head injured patients were admitted in our hospital. Among these patients, seventeen patients with radiological evidence of primary brain stem lesion were included in the study. Inclusion criteria are head injured patients with positive brain stem intensity change in MR imaging and negative hemorrhagic lesion such as epidural or subdural hematoma on CT. The clinical symptoms such as level of consciousness or cranial nerve palsy were not included in this criteria of brain stem lesion. Those who presented cerebral herniation associated with secondary brain stem injury were excluded. Some patients showed dilated pupil or imaging evidence of supratentorial masses. All these patients with dilated pupil or supratentorial masses, however, were confirmed not to have transtentorial herniation or brain stem compression by MRI. Some patients had supratentorial mass lesions, 2 of basal ganglia hemorrhage and 2 of subdural hematoma. All these patients

with supratentorial masses received the operation of hematoma drainage. Intracranial pressure(ICP) monitor was performed for 7 patients including the patients with supratentorial mass lesions. Only one patient with putaminal hemorrhage revealed elevated ICP about 30 mmHg in day 4. This patient did not showed the radiological evidence of transtentorial herniation and the diagnosis of traumatic brain stem injury was made on day 2 with MRI. Other 6 patients with ICP monitor showed normal ICP during the monitoring. Initial neurological status was evaluated with the Glasgow coma scale(GCS)(15). Patients were classified into two outcome groups, those with good outcome and those with a poor outcome. The good outcome group included patients with or without mild disability. The poor outcome group included patients who were severely disabled, in a vegetative state or dead.

Radiological study

The CT scan was carried out on admission in all cases. MRI was carried out within 2 days of the injury in 12 cases. In 5 cases MRI was performed on day 4 to 6 after the injury. Continuous monitoring of electrocardiograph, blood pressure and manual or mechanical ventilation has been continued during MRI examination (7). MRI was performed using a Toshiba MRT-50A with a 0.5 Tesla magnetic field strength.

Images were obtained in continuous 10 mm slices in transaxial planes using the sequence of a repetition time(TR) of 300 msec and a echo delay time(TE) of 14 msec as T1 weighted image, and the sequence of a TR of 2000 msec and a TE of 80 msec as T2 weighted image.

RESULTS

Neurological findings

Table 1 shows the age, gender, GCS score on admission, neurological findings on admission and outcome score at discharge. All the patients except one sustained injury in car accidents. Patients' age ranged from 16 to 75 years with mean age of 35.5 years. There were 10 male and 7 female patients. The GCS score was less than 8 in twelve patients (70.6%). Oculomotor nerve palsy or anisocoria was observed in 10 patients (58.8%) and trochlear nerve palsy was seen in 2 patients. One patient died from sepsis 4 months after the injury.

Final outcome was judged as good in 11 patients and poor in 6 patients. All patients with a GCS score over 12 had a good outcome. Among 12 patients with GCS score below 9, the mean GCS score on admission was 5.4 for patients with a good outcome and 5.2 for those with a poor outcome, so that there was no significant difference in GCS score at

admission. There was no significant difference in focal neurological findings between two groups at the time of admission. Two patients with trochlear nerve palsy showed complete recovery. Among the 4 patients with oculomotor nerve palsy, 2 patients showed no improvement and 2 patients showed improvement of eye movement but anisocoria remained.

Radiological findings

Table 2 summarizes MRI findings of all patients. MRI revealed midbrain injury in 14 among 17 cases. Pontine lesions were seen in 6 patients. Corpus callosum injury was demonstrated in 3 patients of our patients. All brain stem injuries were revealed as high intensity areas on T2 weighted images while only 2 lesions were demonstrated as low intensity areas on T1 weighted images.

Figures 1 and 2 show a schematic depiction of brain stem injury on the basis of MRI T2 weighted images. Dorsal midbrain lesions and dorsal pontine lesions were predominantly seen in the poor prognosis group (Table 3). The good prognosis group had lesions of the ventral midbrain and pons or small lesions of the dorsal midbrain and pons. One patient with a ventral lesion in the poor prognosis group died due to the systemic complications not due to the neurological outcome.

CT scan demonstrated salt and pepper like mixed density areas

which indicated a contusional lesion in the brain stem only in 3 cases and SAH around the brain stem in 12 cases. Concurrent supratentorial lesions were found in 12 cases. CT scan demonstrated 2 dorsolateral midbrain injuries. These 2 patients demonstrated extensive dorsolateral midbrain lesions on MRI and had a poor outcome. Small dorsal brain stem lesions and ventral brain stem lesions which were visible on MRI were not demonstrated on CT scans.

ABR

The ABR was examined in 13 of 17 patients and 3 patients revealed ABR abnormalities. Two patients showed unilateral low voltage or no response and the other showed bilateral prolongation of the latency after the third wave. These 3 patients remained severely disabled. All the 10 patients who showed normal ABR had a good recovery except for one patient who died due to sepsis in the chronic phase.

Correlation of neurological and radiological findings

The patients with lesions involving the superior colliculus or cerebral peduncle lesion demonstrated initial GCS score of 12 or higher. The patients with lesions involving the dorsolateral midbrain, cerebellar peduncle and pons showed initial GCS score of 8 or less. Focal neurological symptoms correlated well with brain stem lesions on MRI.

Patient with oculomotor nerve palsy had an ipsilateral dorsolateral midbrain lesion on MRI.

DISCUSSION

Prognostic factor of brain stem lesion

Our results indicated that a diffuse dorsolateral brain stem lesion on MRI and an abnormal ABR were factors of poor prognosis. While a focal brain stem lesion on MRI and a normal ABR were factors of good prognosis (8). Recent study showed the mortality of the patient with bilateral brain stem lesion on MRI was high and that with unilateral lesion was low (3). The percentage of cases with concomitant supratentorial intracerebral hematoma was more than double in the patient with bilateral brain stem lesion comparing with that in the patient with unilateral brain stem lesion, and they did not discuss the size and location of brain stem lesion on MRI. However, these results demonstrated the patient with diffuse brain stem lesion has poor prognosis.

Rosenblum et al. reported that at successively low intracranial pressure (ICP) the identification of a midbrain lesion by evoked potentials is a more reliable predictor of the prognosis in head injured patients than the ICP level (13). The combination of MRI findings and ABR in the

acute stage may provide more reliable information in regard to the prognosis. Our previous study showed that abnormal ABR means no reversibility of disturbance of consciousness, but normal ABR does not mean good recovery (8). The sensitivity of ABR to detect brain stem dysfunction seem to be low. Our study population is too small to draw definite conclusion and the follow up and determination of final outcome were not enough, so prospective study for large patients with head injury should be designed in near future.

Diagnostic criteria for brain stem injury

The term “primary brain stem injury” was first described by Mitchell and Adams in 1973, and they concluded primary brain stem injury does not occur in isolation but is only one component of diffuse damage of the white matter throughout the brain (9). Immediate and sustained loss of consciousness following the injury and abnormal brain stem reflexes were the clinical criteria for the diagnosis of the primary brain stem injury (6). However, with the advent of CT scan and MRI, it became possible to study pathological changes in the brain stem while patients are alive. The superior sensitivity of MRI over CT in detecting the lesion and anatomical delineation of the lesion in patients with intracranial pathology has been well documented, especially in case of patients with a nonhaemorrhagic

lesion or brain stem lesion (5). At present, MRI is the most reliable examination for the diagnosis of primary brain stem injury and may depict subclinical abnormality.

Mechanism of pathogenesis of primary brain stem injury

Mitchell and Adams concluded that primary brain stem injury is always accompanied by supratentorial cerebral diffuse axonal injury (9). However, in our series MRI revealed more mild forms of brain stem injury. Based on the results of the present study, clinical and radiological diagnosis of primary brain stem injury without severe diffuse axonal injury has been demonstrated. Gennarelli et al. demonstrated that axonal injury in the cerebral white matter and brain stem was produced by coronal head acceleration and suggested that primary brain stem injury results from shear strain due to rotational acceleration (4). This hypothesis can not explain primary brain stem injury without severe diffuse axonal injury as seen in our cases. Another plausible mechanism is direct impact of brain stem on the free edge of the tentorium (2,11). In our MRI study, brain stem lesions in patients with good prognosis located at the lateral side of the midbrain-pontine junction, which is the nearest point between the tentorial edge and the brain stem (14). The lesions in patients with good prognosis seemed to be a direct focal brain stem injury caused by the

impact against the tentorial edge. On the other hand, dorsal brain stem lesions in patients with poor prognosis were thought to be due to shear strain, therefore these patients sustained a brain stem injury as part of a diffuse axonal injury and often presented with prolonged coma. MRI may distinguish these two subgroups, brain stem injury with mild DAI and focal brain stem injury, for which the clinical outcome is quite different.

CONCLUSION

MRI, especially T2 weighted image in the acute stage, proved to be the most sensitive and specific diagnostic means in case of primary brain stem injury. According to MRI findings, primary brain stem injuries can be divided into two subgroups, one is a direct focal brain stem injury due to impact against the tentorial edge and the other is a brain stem injury due to shear strain associated with diffuse axonal injury. It is important to differentiate these two subgroups, because the former tends to have better prognosis than the latter.

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LEGENDS FOR FIGURES

Fig.1 Schematic depiction of primary brain stem lesions in the good prognosis group on the basis of MRI T2 weighted image.

Fig.2 Schematic depiction of primary brain stem lesions in the poor prognosis group on the basis of MRI T2 weighted image.

Table 1 Summary of cases

Case	age	sex	GCS	Neurological findings	outcome score
good outcome					
1	50	F	14	trochlear nerve palsy	GR
2	21	M	8	oculomotor nerve palsy	GR
3	19	F	5	coma, anisocoria, tetraparesis	GR
4	16	M	7	coma, hemiparesis	GR
5	35	M	4	coma, anisocoria, hemiparesis	GR
6	48	F	12	anisocoria	GR
7	28	M	3	coma, hemiparesis	GR
8	19	M	14	normal	GR
9	55	M	15	double vision	GR
10	24	F	7	coma	GR
11	19	F	4	coma, oculomotor nerve palsy	GR
poor outcome					
12	75	M	9	anisocoria, hemiparesis	SD
13	34	M	3	coma, trochlear nerve palsy, hemiparesis	SD
14	73	F	6	coma, oculomotor nerve palsy	SD
15	20	F	4	coma, oculomotor nerve palsy	SD

16	47 M	6	coma, anisocoria, hemiparesis	D
17	20 M	3	coma	SD

GCS: Glasgow coma scale

GR: good recovery

SD: severely disabled

D: dead

Table 2 MRI findings

Case MRI findings

midbrain injury

1	SC(T2)
2	dorsolateral MB(T2)
3	SC, ventral Po, CC(T2)
4	ventral MB(T2)
6	CP(T2)
8	CP(T1,T2)
9	CP(T2)
10	dorsal Po MB(T2)
11	Po CP(T2)
12	dorsolateral MB(T2)
14	dorsal MB(T2)
15	dorsolateral MB(T2)
16	CP, Po(T2)
17	dorsal MB, CC(T2)

pontine injury

5	ventral Po, CC(T2)
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- 7 dorsal Po(T2)
13 cerebellar peduncle(T1,T2)

CP: cerebral peduncle

MB: midbrain

SC: superior colliculus

CC: corpus callosum

Po: pons

T1: low intensity lesion on T1 weighted images

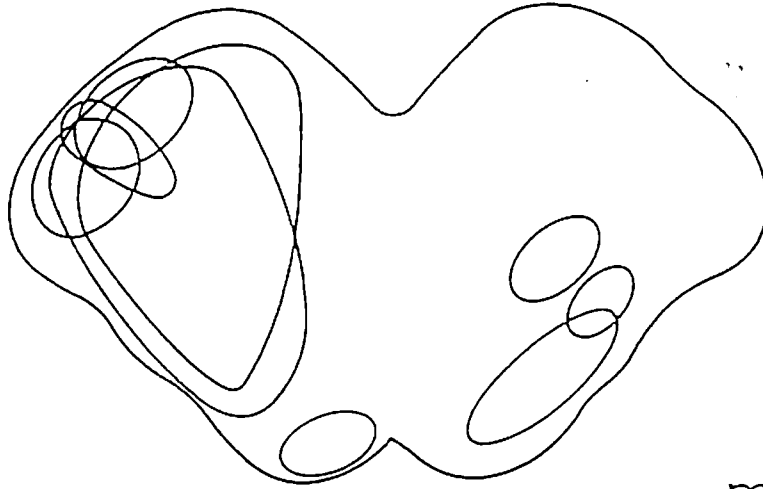
T2: high intensity lesion on T2 weighted images

Table 3 Large dorsal brain stem injury and prognosis

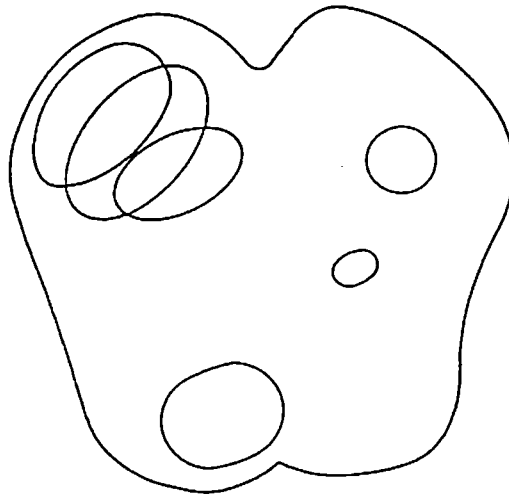
dorsal brain stem injury	good outcome	poor outcome
positive	3	5
negative	8	1

The difference is significant in Chi-square test ($p < 0.05$).

good prognosis group

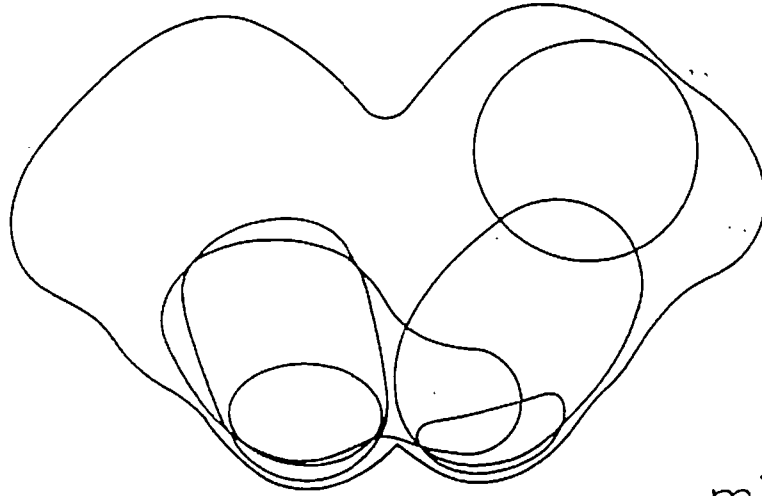


midbrain

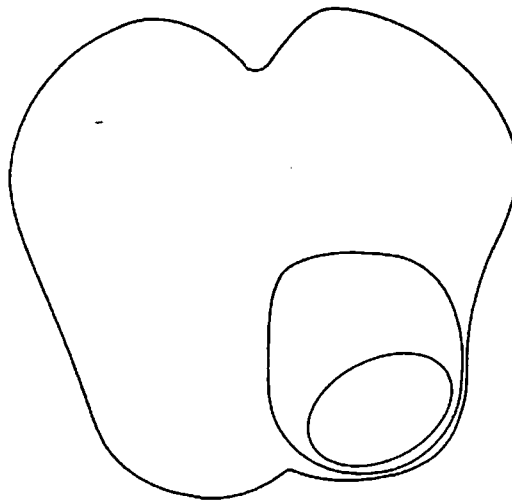


pons

poor prognosis group



midbrain



pons